NICOLAUS COPERNICUS UNIVERSITY IN TORUŃ

Journal of Education, Health and Sport. eISSN 2391-8306.

Journal Home Page
https://apcz.umk.pl/JEHS/index

JAROWICZ, Monika, MARCZAK, Zuzanna, SITO, Maksymilian, JURKOWSKI, Filip, WÓJCIK, Łukasz, GAWRON, Natalia, BOJANOWSKI, Hubert Tomasz, DOBRACKA, Aleksandra, ZACHAR, Kinga and CZMYR, Jędrzej. Sweat in Skin Homeostasis and Dermatological Disorders in Athletes: Structural, Functional, and Clinical Perspectives - A Narrative Review. Journal of Education, Health and Sport. 2026;91:70646. eISSN 2391-8306. https://doi.org/10.12775/JEHS.2026.91.70646

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 07.04.2026. Revised: 04.05.2026. Accepted: 05.05.2026. Published: 10.05.2026.

Sweat in Skin Homeostasis and Dermatological Disorders in Athletes: Structural, Functional, and Clinical Perspectives- A Narrative Review

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ABSTRACT

Introduction and purpose. Sweat is essential for skin homeostasis. In physically active individuals, increased sweating supports protective skin functions but may also trigger dermatological conditions. This review summarizes current knowledge on sweat gland types, their functional diversity, and the role of sweat in skin homeostasis, dermatoses, and gland disorders.

Material and method. A narrative review of the literature was conducted using PubMed and Google Scholar, including original articles and reviews on sweat gland physiology, sweat composition, and related dermatoses.

Results. Secretions from eccrine, apocrine, and apoeccrine glands contain water, electrolytes, and bioactive molecules that contribute to epidermal barrier integrity and immune functions. In physically active individuals, increased sweating improves heat dissipation, enhances stratum corneum hydration, and facilitates the delivery of antimicrobial and immunomodulatory factors to the skin surface. However, prolonged moisture and friction can disrupt the skin barrier, promoting microbial overgrowth and contributing to cutaneous irritation, infections, and the exacerbation of dermatoses. Sweat gland disorders, further illustrate the consequences of dysregulated sweating, leading to impaired thermoregulation, inflammation, discomfort, and reduced physical performance.

Conclusions. Sweat has multifactorial effects on skin health, influenced by environmental and individual factors. Understanding sweat physiology and related disorders is essential for preventing and managing skin diseases.

Keywords: sweat physiology, sweat glands, skin homeostasis, epidermal barrier, sweat-related disorders, athletes

1. Introduction

The skin is the largest organ in the human body, playing a fundamental role in maintaining homeostasis and protecting against environmental agents. Its function relies on complex regulatory mechanisms, one of which is the activity of sweat glands (1). Sweat plays a key role in thermoregulation and maintaining the proper hydration of the stratum corneum (2, 3). It also contributes to the regulation of skin pH and the composition of the skin microbiome. Sweat, composed of water, electrolytes, and biologically active compounds, helps maintain the balance of the skin microenvironment. It supports proper epidermal barrier function, reduces transepidermal water loss, and protects the skin against irritants and microorganisms (4). The importance of properties of sweat is particularly evident in physically active individuals. In athletes, intense and often prolonged exercise leads to increased sweat gland activity, resulting in greater skin exposure to moisture and temperature fluctuations. Simultaneously, the use of sports clothing and equipment increases exposure to mechanical factors such as friction and pressure. These conditions may act as both a stimulus for physiological skin adaptation and a factor that can disrupt skin homeostasis. Consequently, beyond its primary physiological role, sweat may indirectly contribute to impairment of the skin barrier, alterations in the microbiome, and the development of inflammatory processes (5). Excessive or prolonged exposure to sweat is associated with an increased susceptibility to bacterial and fungal infections, such as dermatophytosis and trichomycosis axillaris (6, 7). Sweat can also exacerbate atopic dermatitis, intertrigo, and dyshidrotic eczema (8-10). Furthermore, sweat-related dermatological diseases are a group of conditions involving abnormal sweat gland and duct production, retention, or inflammation. Miliaria, hyperhidrosis, anhidrosis, hidradenitis suppurativa, and Fox-Fordyce disease may lead not only to cutaneous symptoms but also to systemic dysregulation, which directly impacts physical activity, mental and physical health, and quality of life (11-15). This review aims to systematise the current knowledge on sweat gland types and their structural and functional diversity. It also analyses the role of sweat in maintaining skin homeostasis, its influence on the development and progression of dermatological disorders, and the significance of sweat gland dysfunction in physically active populations.

2. Types of Human Sweat Glands and Their Structural and Functional Differences

Human skin is a complex organ that plays a key role in maintaining homeostasis, particularly in thermoregulation and excretion (16). These processes involve three main types of sweat glands: eccrine, apocrine, and apoeccrine, which differ in their structure, distribution, and physiological roles (4).

Eccrine sweat glands are the most numerous and widely distributed type, with an estimated 2-4 million glands present across the body. Their distribution is uneven across the body, with the highest density occurring on the palms and soles (17). Structurally, the eccrine gland is composed of a coiled secretory portion located in the deep dermis and a duct that extends toward the epidermis, where it forms the intra-epidermal segment known as the acrosyringium. The secretory portion contains clear cells responsible for producing primary sweat, dark cells that contain numerous granules and may store bioactive substances, and myoepithelial cells that assist in the expulsion of sweat (18). The duct, formed by basal and luminal epithelial cells, modifies the primary secretion by reabsorbing sodium and chloride ions, resulting in the release of hypotonic sweat onto the skin surface. Eccrine glands primarily contribute to thermoregulation and are mainly activated by thermal stimuli through sympathetic cholinergic pathways, although they may also respond to emotional and exercise-related stimulation (4). The secretion produced by these glands consists predominantly of water and sodium chloride, together with small amounts of minerals, metabolites, and other compounds derived from interstitial fluid (19).

Apocrine sweat glands differ from eccrine glands in terms of their morphology and function. They are larger and open into hair follicles rather than directly onto the skin surface. They are found mainly in hairy areas of the body, particularly in the axillary, mammary, perineal, and genital regions, as well as on the scalp and face. Although apocrine glands are present from birth, their secretory activity begins at puberty. The secretory portion forms a relatively large coiled structure composed primarily of secretory and myoepithelial cells. Secretion occurs through a characteristic “decapitation” mechanism, in which portions of the apical cytoplasm are released into the gland lumen (20). The resulting secretion is a viscous, lipid-rich fluid that is initially odorless but may contribute to body odor after bacterial decomposition. Apocrine glands are considered to function as scent glands and may play a role in pheromone-related signaling, although this role is limited in humans (21). These glands primarily respond to emotional stimuli such as stress, anxiety, pain, or sexual arousal, and their activity is regulated by both adrenergic and cholinergic mechanisms (22).

A third type, the apoeccrine sweat gland, shows features intermediate between eccrine and apocrine glands. These glands develop from eccrine precursors during childhood, typically between 8 and 14 years of age, and are found mainly in the axillary region. Morphologically, they are intermediate in size and consist of a tubular structure with irregularly shaped cells, including eccrine-like and apocrine-like secretory cells, as well as myoepithelial cells. Apoeccrine glands open directly onto the skin surface and produce a continuous watery secretion with a composition similar to eccrine sweat (23). The glands have a high secretory capacity and may account for a substantial proportion of axillary sweating, particularly during adolescence and adulthood. Although their exact physiological role is not fully understood, they respond to both cholinergic and adrenergic stimulation, indicating complex neural regulation (23, 24).

The composition of human sweat varies depending on the type of gland and the stimuli that trigger secretion. Eccrine sweat is mainly composed of water and sodium chloride, but it also contains minerals such as potassium, calcium, and magnesium, trace elements like iron, zinc, and copper, metabolites including lactate, urea, and ammonia, bicarbonate, as well as small amounts of glucose (4). In addition, eccrine sweat contains proteins, antibodies (e.g. IgG, IgA, IgE), antimicrobial peptides (e.g. dermcidin, cathelicidin, lactoferrin), amino acids, cytokines (e.g. IL-1α, IL-1β, IL-6, IL-8, IL-31, tumor necrosis factor α, and transforming growth factor β), proteolytic enzymes, vitamins, and persistent organic pollutants such as organochlorine pesticides or bisphenol A (4, 25-29). Its composition is influenced by factors such as diet, hydration, metabolic rate, health status, drug use, and the body region from which it is secreted. In contrast, apocrine glands produce a viscous secretion that is rich in lipids and also contains proteins, steroids, and the same minerals and metabolites found in eccrine sweat. Comparative studies show that apocrine glands excrete higher levels of urea, sodium, and potassium than eccrine glands, particularly during exercise, indicating that sweat composition depends on both gland type and the triggering factor (30).

3. The Role of Sweat in Skin Homeostasis

Sweating plays a critical role in regulating body temperature. The primary physiological function of eccrine sweat production is heat dissipation: evaporation of sweat from the skin surface removes large amounts of heat, enabling the body to maintain a stable internal temperature during thermal stress and physical exercise. During high-intensity training, increased metabolic activity generates additional heat that stimulates thermoreceptors in the skin and core, signaling the hypothalamus to enhance sweat gland activity. This exercise-induced sweating is essential for preventing hyperthermia and maintaining performance, while also interacting with cardiovascular adjustments and fluid-electrolyte balance (2). In addition, the composition of sweat supports the maintenance of the skin's hydrolipid barrier. Sweat contains water, electrolytes, amino acids, and small amounts of lipids that help maintain proper hydration of the stratum corneum (4). Increased sweating enhances the delivery of these components to the skin surface, which can temporarily improve hydration and support the structural integrity and elasticity of the epidermal barrier despite the additional mechanical and thermal stress associated with sports activities. Natural moisturizing factors (NMF) are low-molecular-weight hygroscopic compounds present in the stratum corneum that play an essential role in maintaining skin hydration by binding and retaining water. NMF consists mainly of amino acids and their derivatives, such as pyrrolidone carboxylic acid, urocanic acid, as well as lactate, urea, citrate, and sugars. Many of these compounds are generated through the degradation of filaggrin. In addition, sweat delivers lactate, urea, and electrolytes to the outer layers of the stratum corneum. Through this mechanism, sweat directly contributes to the NMF pool in the outermost layers of the skin (3). Studies have shown that the onset and increase of sweating during physical activity lead to a measurable rise in stratum corneum hydration, which may persist even after exercise (3, 5). This enhanced hydration, together with the formation of a continuous surface hydrolipid film, contributes to the regulation of transepidermal water loss (TEWL), which is the passive diffusion of water from the deeper layers of the skin to the external environment (31). When the hydrolipid barrier is intact, TEWL remains at a physiological level, preventing excessive skin dehydration (32). Exercise-induced sweating affects the skin acid-base balance. The acidity of sweat is mainly determined by lactic acid and volatile acids such as acetic acid. Although sweat may become temporarily alkaline due to ammonia produced by the skin microbiota, this effect is transient, as ammonia rapidly evaporates and the concomitant increase in lactic acid restores the acidic pH of the skin. As a result, skin surface pH decreases at the onset of sweating, increases during prolonged or intense sweating, and gradually returns toward baseline after exercise, reflecting dynamic changes in the skin acid-base balance (33). Beyond hydration and pH regulation, eccrine sweat contains a diverse array of bioactive molecules that support microbial homeostasis and innate immunity. These components include antimicrobial peptides such as dermcidin, cathelicidin, and lactoferrin, antibodies (e.g., IgG, IgA), cytokines (e.g., interleukins, TNFα), proteolytic enzymes, and micronutrients such as vitamins and trace minerals (4, 25, 29, 34). Dermcidin is constitutively expressed in eccrine sweat glands and is secreted as a full-length precursor. This precursor is then processed into peptides with antimicrobial and antioxidant properties, which provide continuous protection against a broad range of microorganisms, including Staphylococcus aureus, Escherichia coli, Enterococcus faecalis, and Candida albicans. In addition, dermcidin and its derivatives can modulate innate immune responses by influencing cytokine production, thereby contributing to both skin defense and immune regulation (35). Other sweat-derived peptides and cytokines modulate local immune responses, while proteolytic enzymes activate or process antimicrobial molecules at the skin surface. Vitamins acting as antioxidants help protect skin cells from oxidative stress and support tissue repair. Increased sweating during exercise enhances the transport of these molecules to the skin surface, thereby supporting the biochemical integrity of the epidermal barrier, even under prolonged moisture exposure and mechanical stress (4).

4. **The Impact of Sweat on Dermatological Conditions in Athletes**

Physical exertion increases sweat production, which in healthy individuals can support the skin's natural protective function. However, sweating can exacerbate symptoms and contribute to increased irritation, inflammation, and disease severity in certain dermatological conditions. In atopic dermatitis, patients often exhibit reduced sweat production and delayed onset of sweating, resulting in functional anhidrosis. This impairment arises from ductal obstruction by keratin plugs, intrinsic defects in sweat gland secretion, autonomic dysfunction, and histamine-mediated inhibition of perspiration (36). In atopic dermatitis, the sweat itself can also exacerbate skin lesions through multiple mechanisms involving barrier dysfunction, inflammation, and microbial imbalance (8). Although sweat normally reaches the skin surface without affecting viable keratinocytes, abnormalities in sweat gland function and duct integrity in atopic dermatitis allow sweat components to penetrate surrounding tissues (36). Sweat contains proinflammatory molecules, including IL-1α, IL-1β, IL-31, and IL-8, which activate keratinocytes and induce cutaneous inflammatory responses. Exposure of cultured human keratinocytes to sweat leads to activation of key signaling pathways, including NF-κB, ERK, and JNK, accompanied by decreased IκBα levels (26). Heat and moisture associated with sweating further promote vasodilation and stimulate peripheral nerve endings, intensifying pruritus. Moreover, elevated glucose levels in sweat observed in these patients may negatively affect epidermal barrier repair (37). Alterations in skin surface pH and increased colonization by microorganisms such as Staphylococcus aureus further contribute to inflammation (38). Clinically, these processes manifest as intensified itching, burning sensation, erythema, and worsening eczematous lesions, often accompanied by scratching that perpetuates the inflammatory cycle (38, 39).

Dermatophytosis is a superficial fungal infection of keratinized tissues caused by dermatophytes, including Trichophyton, Microsporum, and Epidermophyton, which utilize keratin as a nutrient source (40). Predisposition to dermatophytosis is influenced by a combination of host and environmental factors, including occlusive clothing, inadequate hygiene, metabolic disorders such as diabetes mellitus, immunosuppression, genetic susceptibility, and socioeconomic conditions. Individuals exposed to frequent skin-to-skin contact, such as athletes, are also at increased risk. Additionally, increased moisture and temperature associated with sweating contribute to maceration of the stratum corneum, weakening the epidermal barrier and facilitating fungal penetration and spread (6). These conditions favor fungal persistence and may exacerbate infection, leading clinically to intensified erythema, scaling, and pruritus, often presenting as well-demarcated lesions with active borders (41).

Intertrigo is an inflammatory dermatosis that arises within opposing skin folds, where friction, occlusion, and moisture accumulation compromise the epidermal barrier. Its pathogenesis is driven by mechanical irritation and maceration of the stratum corneum, which facilitates penetration and overgrowth of opportunistic microorganisms, such as Candida, Corynebacterium minutissimum, and group A beta-hemolytic streptococcus (9, 42). Sweat plays a significant role in both the initiation and exacerbation of lesions, as persistent moisture increases skin hydration, disrupts barrier function, and creates a warm, humid microenvironment conducive to microbial proliferation (9). In athletes, these processes are intensified by prolonged physical activity, repetitive motion, tight clothing, and increased sweating, which together enhance friction and delay evaporation, thereby predisposing to more severe or recurrent forms of intertrigo (42). Clinically, the condition manifests as well-defined erythematous patches that are often moist, shiny, and prone to erosion (43). In more advanced cases, fissuring, exudation, and satellite lesions may appear, particularly in the presence of fungal infection. Symptoms typically include pruritus, burning, pain, and discomfort, often worsening during continued activity or heat exposure (42).

Trichomycosis axillaris is a superficial bacterial infection of the hair shaft, most commonly affecting axillary hair, and is caused by aerobic Corynebacterium species. Its pathogenesis is related to the colonisation of the hair shaft by bacteria and the formation of pigmented concretions composed of microorganisms and altered sweat components. This process is facilitated by local microenvironmental changes and disturbances in apocrine sweat production (7). Excessive sweating plays a central role by increasing moisture and promoting bacterial proliferation, adhesion, and biofilm-like sheath formation around hairs (44). Other key risk factors include hot and humid climates, obesity, and hygienic neglect, which, together with intense physical activity, occlusive clothing, and repeated friction, make athletes particularly susceptible to trichomycosis axillaris development (7). Clinically, the condition presents with yellow, white, red, or black nodular concretions firmly adherent to hair shafts, sometimes accompanied by malodor (bromhidrosis) or colored sweat (chromhidrosis), although it may remain asymptomatic. The condition is generally benign, but it can lead to cosmetic concerns, hair fragility, as well as causing a misdiagnosis and delay appropriate treatment (7, 45).

Dyshidrotic eczema is an inflammatory vesicular skin eruption that primarily affects the palms and soles and may occur in athletes exposed to prolonged, excessive sweating, contact allergens, irritants, and smoking (10). While the exact pathomechanism of dyshidrotic eczema is unclear, it is associated with immune dysregulation, as demonstrated by the significant presence of CD3, CD8, CD45, anti-MPO, anti-human fibrynogen, and anti-human IgE antibodies in affected areas (46). The condition is characterized by vesicles with a "tapioca pudding" appearance on the lateral aspects of the fingers, which may enlarge to form bullae and are often accompanied by intense itching. In athletes, these symptoms can lead to significant discomfort and impaired training performance, as well as an increased risk of secondary infection due to skin barrier disruption (10).

Regular and attentive skin care is essential for athletes, as it helps mitigate the adverse effects of sweat and excess moisture on the skin barrier, while also preventing or alleviating exercise-related dermatoses. Following intense physical activity, the skin should be cleansed promptly using gentle products to remove sweat and microorganisms, thereby reducing irritation and minimizing the risk of infection. Wearing dry, breathable, moisture-wicking clothing, along with frequently changing damp garments, is equally important. These measures help limit the proliferation of fungi and bacteria in skin folds and interdigital spaces, reducing maceration and mechanical damage to the skin (5). Consequently, they lower the risk of infections and inflammatory conditions such as trichomycosis axillaris, dermatophytosis, and intertrigo. In individuals with atopic dermatitis, particular attention should be paid to the prompt removal of sweat following exercise and to the regular application of emollients. These practices support the restoration of the skin barrier, enhance hydration, and may help alleviate inflammatory symptoms (36, 47).

5. Sweat Gland Disorders and Their Repercussions in Sports

Sweat-related dermatological diseases are conditions associated with abnormal production, retention, or inflammation of sweat glands and sweat ducts. The most common disorders include miliaria, hyperhidrosis, anhidrosis, hidradenitis suppurativa, and dyshidrotic eczema.

Miliaria, although most commonly observed in neonates due to the immaturity of their eccrine sweat glands, can also occur in athletes, particularly during exercise in hot and humid environments when excessive sweating occurs. The pathomechanism involves temporary obstruction of the sweat ducts, which prevents normal sweat secretion and leads to retention of sweat within the skin. This results in the formation of vesicles, pustules, or inflammatory lesions (48, 49). Clinical presentation depends on the depth of duct blockage. Miliaria crystallina appears as small, clear vesicles within the stratum corneum (48). Miliaria rubra is characterized by pruritic, erythematous papules in the mid-epidermis; when these papules progress to pustules, the condition is termed miliaria pustulosa (11, 50). Miliaria profunda presents as large, firm papules caused by obstruction of eccrine ducts at the dermal-epidermal junction, which may manifest with pruritus. (50). In athletes, this condition may cause discomfort, itching, and reduced training performance, and it can increase the risk of secondary skin infections. Management of miliaria aims to reduce sweating and prevent blockage of eccrine ducts. General recommendations include keeping the environment cool, wearing loose and breathable clothing, minimizing skin occlusion, and managing underlying conditions, such as fever (11). Miliaria crystallina usually resolves spontaneously, whereas miliaria rubra may require short-term topical corticosteroid therapy (48). In cases of miliaria pustulosa, topical antibiotics (e.g., clindamycin) are indicated to treat secondary bacterial infection. Data on miliaria profunda are limited, however, some evidence suggests potential benefit from combined therapy with oral isotretinoin and topical anhydrous lanolin (11).

Hidradenitis suppurativa is a chronic, recurrent inflammatory skin disorder characterized by painful nodules, abscesses, cysts, and draining dermal sinuses that heal with scarring. It predominantly affects apocrine gland-rich areas, including the axillae, inguinal and anogenital regions, and the breasts in women. Patients commonly report pain, pruritus, malodor, localized warmth, and burning, which may considerably limit physical activity due to discomfort, skin tightness, restricted movement, and increased susceptibility to friction-related exacerbation during training. Hidradenitis suppurativa is driven primarily by follicular occlusion, which triggers a cascade of immune-mediated inflammation involving neutrophils, lymphocytes, and proinflammatory cytokines such as TNF-α, IL-1β, IL-17, and IL-23. Genetic factors such as γ-secretase gene mutation, dysregulation of the Notch signaling pathway, and hormonal influences further contribute to disease progression (12). In addition, environmental triggers such as mechanical pressure and friction promote follicular occlusion and may result in the rupture of distended hair follicles (51). Management of hidradenitis suppurativa includes both pharmacological and surgical approaches, depending on disease severity and clinical staging. General measures such as weight reduction, hygiene, and topical therapies may help reduce symptoms, while systemic treatments, including antibiotics, retinoids, hormonal agents, and immunosuppressants are commonly used for disease control, although their long-term efficacy remains limited. Biologic therapies targeting inflammatory pathways have shown promising results but require careful consideration of potential adverse effects. In advanced stages, surgical intervention, particularly wide excision of affected tissue, remains the most effective method for reducing recurrence and achieving long-term disease control (52).

Hyperhidrosis is a condition characterized by excessive sweating beyond thermoregulatory needs and is classified as primary or secondary. Primary hyperhidrosis is more common and typically results from idiopathic overactivity of the sympathetic nervous system. It most often affects focal areas such as the palms, soles, axillae, and face. (53). In contrast, secondary hyperhidrosis is usually generalized and occurs as a consequence of underlying conditions, including endocrine, metabolic, infectious, or neurological disorders, as well as certain medications (54). Hyperhidrosis is associated with psychological distress, including anxiety and depression, which has a significant impact on both personal and professional life, leading to impaired social functioning, reduced work or academic performance, and deterioration in overall quality of life (13, 55). In addition, the condition may reduce participation in physical activity and training efficiency. Among athletes, hyperhidrosis can contribute to discomfort, impaired grip, skin maceration, and increased susceptibility to infections (13, 56, 57). Treatment of hyperhidrosis is individualized and depends on the severity and location of sweating, comorbidities, and patient tolerance. First-line therapy for localized disease includes topical agents such as aluminum salts and topical anticholinergics, while systemic therapy with glycopyrrolate or oxybutynin may be used off-label when topical treatment is ineffective. Botulinum toxin injections remain a highly effective treatment for focal hyperhidrosis by inhibiting acetylcholine release from the nerve endings, although repeated treatments may be required and may be associated with transient discomfort (58, 59). Device-based treatments for hyperhidrosis act either by damaging sweat glands (e.g., fractionated microneedle radiofrequency, micro-focused ultrasound, microwave thermolysis) or by altering their function (iontophoresis). Procedures that destroy glands may offer more prolonged effects, although their efficacy is supported by low-quality evidence. Surgical management, including liposuction curettage, demonstrates moderate effectiveness with sweat reduction of 30–80% lasting at least 6 months. In contrast, although iontophoresis can achieve a 30–90% reduction in sweating, its effects are transient and require ongoing, regular treatments to maintain clinical benefit (60).

Anhidrosis is a condition characterized by localized or generalized failure of sweat production in response to thermal or chemical stimuli, often presenting with dry skin, heat intolerance, tendency to overheat, hyperemia, and previous faintness during physical activity (61). In athletes, anhidrosis significantly increases the risk of heat-related illnesses, such as heat exhaustion or heat stroke, and may severely limit exercise capacity and safety during training (14). Anhidrosis may be primary (idiopathic) or secondary to a wide range of underlying conditions. Central and neuropathic causes include lesions affecting the brain, spinal cord, or autonomic nerves, as well as systemic diseases, genetic syndromes, and medications that impair neural transmission. Peripheral forms result from structural or functional abnormalities of the sweat glands, including genetic disorders, tissue destruction, or obstruction due to dermatologic conditions. The management of anhidrosis is determined by its underlying etiology, however, in all cases, patients should be protected from overheating by providing a cool environment (62). Steroid pulse therapy represents the most frequently reported treatment for acquired idiopathic generalized anhidrosis, providing symptomatic improvement in over half of patients. The recurrence rate is approximately 43–48%, therefore, early initiation, appropriate seasonal timing, and careful monitoring are recommended to maximize treatment effectiveness and minimize adverse effects (63). The management of secondary anhidrosis focuses on eliminating the causative factor, as improvement of the underlying disease or discontinuation of the triggering medication often results in restoration of normal sweating (62).

Fox-Fordyce disease is a rare, chronic inflammatory disorder primarily affecting apocrine gland-bearing areas, most commonly seen in adolescent and young adult women. Its exact cause remains unclear, but potential triggers include hormonal influences or local trauma, such as from laser hair removal. The pathophysiology involves obstruction of the apocrine duct by a keratin plug, leading to retention of secretions, rupture of the gland, and secondary dermal inflammation (64). Fox-Fordyce disease manifests clinically as multiple, firm, folliculocentric papules that are typically skin-colored, yellowish, or slightly brown. It most commonly affects the axillae, although the pubic, periareolar, and thoracic regions may also be involved (65, 66). Patients frequently report pruritus with chronic scratching leading to lichenification. Heat, humidity, and friction can exacerbate pruritus and irritation, potentially impairing participation in sports and other physically demanding activities (67). Additionally, hair loss can occur in the affected areas, while anhidrosis is rare (15). Histopathologically, Fox-Fordyce disease is characterized by perifollicular lymphocytic infiltrates and the presence of foamy histiocytes. Additional features include hyperkeratosis, acanthosis, and spongiosis, as well as infundibulum dilation and obstruction of apocrine sweat glands (66). Management remains challenging due to limited high-quality evidence. First-line treatments generally focus on topical or oral agents that reduce inflammation, prevent follicular or ductal occlusion, and alleviate pruritus. These include corticosteroids, retinoids, benzoyl peroxide, clindamycin, calcineurin inhibitors, and oral contraceptives, which are selected for their relative safety and ease of use. Partial improvement is commonly observed, often within weeks of initiation. For more severe or refractory cases, options such as surgical excision, fractional lasers, liposuction-assisted curettage, botulinum toxin injections, and microwave therapy may be considered (15).

Reported treatment outcomes indicate that approximately 70% of patients experience significant improvement, including reduced pruritus, smaller lesion size, hair regrowth, or partial restoration of sweating, whereas complete resolution is more commonly observed during pregnancy, in women after menopause, or following hormonal modulation. Recurrence remains frequent, emphasizing the need for ongoing monitoring and individualized management (64).

6. Conclusions:

Human sweat glands play a fundamental role in skin homeostasis, contributing not only to thermoregulation but also to the regulation of hydration, pH balance, barrier integrity, and cutaneous immune function. Sweat contains water, electrolytes, metabolites, antimicrobial peptides, cytokines, and trace elements, which together support the skin's structural and biochemical defenses. In athletes, who exhibit elevated metabolic heat production and are more frequently exposed to factors predisposing to skin infections, the role of sweat appears to be particularly important. However, under conditions of prolonged moisture, friction, and occlusion, sweating may disrupt epidermal homeostasis, creating a microenvironment conducive to inflammation, microbial overgrowth, and barrier impairment. Atopic dermatitis may be exacerbated by sweat, which can penetrate a compromised skin barrier and trigger inflammatory signaling. Dyshidrotic eczema is associated with immune dysregulation and may also be worsened by excessive sweating. In addition, intertrigo, dermatophytosis, and trichomycosis axillaris, can be promoted by prolonged moisture, friction, and local hyperhidrosis. Abnormalities in sweat gland function, including miliaria, hyperhidrosis, anhidrosis, hidradenitis suppurativa, and Fox-Fordyce disease, further illustrate the clinical consequences of dysregulated sweating. These conditions can lead to pain, pruritus, malodor, impaired thermoregulation, reduced exercise capacity, and diminished quality of life. In athletes, the prevalence of these conditions may be increased due to repetitive mechanical stress, occlusive clothing, and prolonged increased sweat production, which can further aggravate lesions and delay recovery. Effective prevention and management of sweat-related dermatoses require an integrated strategy that combines appropriate hygiene practices, maintenance of skin barrier function, and the use of breathable, moisture-wicking materials. Interventions should be individualized and may include topical or systemic therapies, as well as procedural and surgical approaches. Early recognition and targeted management are particularly important in physically active individuals to prevent progression of lesions and minimize functional impairment. Overall, sweat should be regarded as a multifaceted component of skin physiology. While it plays a crucial role in maintaining epidermal balance and innate defense mechanisms, specific environmental and mechanical conditions may shift its effects toward promoting irritation, inflammation, and microbial imbalance. This duality is especially evident in athletes, in whom increased sweating and external stressors amplify both the protective and detrimental aspects of perspiration. A comprehensive understanding of sweat gland function and related pathologies is essential for developing effective preventive strategies and optimizing dermatological care. Future research that integrates physiological, microbiological, and clinical perspectives will be crucial for advancing targeted interventions and enhancing skin health, physical performance, and overall quality of life in physically active populations.

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Supervision: MJ, ZM, MS, FJ, HB
Project administration: MJ, HB
All authors have reviewed and consented to the publication of the final version of the manuscript.

Disclosure
Funding: No funding was received.
Institutional Review Board Statement: Not applicable.
Informed Consent Statement: Not applicable.
Data Availability Statement: Not applicable.
Acknowledgements: Not applicable.
Conflicts of Interest: The authors declare no conflict of interest.
Declaration of Generative AI and AI-assisted technologies in the writing process: During the preparation of this work, the authors used large language models (e.g. Gemini) in order to improve English language, readability, and text formatting. After using this tool/service, the authors thoroughly reviewed and edited the content as needed and take full and sole responsibility for the final content of the publication.

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